Posterior Tibial Tendon Dysfunction: An Overlooked Cause of Foot Deformity

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Abstract

Posterior tibial tendon dysfunction is the most common cause of adult acquired flatfoot. Degenerative changes in this tendon, lead to pain and weakness and if not identified and treated will progress to deformity of the foot and degenerative changes in the surrounding joints. Patients will complain of medial foot pain, weakness, and a slowly progressive foot deformity. A "too many toes" sign may be present and patients will be unable to perform a single heal raise test. Investigations such X-ray, ultrasound and magnetic resonance imaging will help stage the disease and decide on management. The optimal manage may change based on the progression of deformity and stage of disease. Early identification and prompt initiation of treatment can halt progression of the disease. The purpose of this article is to examine the causes, signs, symptoms, examinations, investigations and treatment options for posterior tibial tendon dysfunction.

Keywords: Adult acquired flatfoot, pes planovalgus, posterior tibial tendon

Introduction

Posterior tibial tendon dysfunction is the most common cause of adult acquired flatfoot disease. The changes of advanced disease are obvious and have significant morbidity associated with it. Tendon degeneration, however, begins far before clinical disease is apparent.[1] By detecting early posterior tibial tendon disease, progression may be halted with nonoperative means; if left to progress, surgical reconstruction with osteotomy and arthrodesis becomes necessary.[2] The purpose of this article is to review the pathphysiology, clinical stages, investigations and treatment options for posterior tibial tendon dysfunction, so that general practitioners can easily identify early disease and initiate treatment before deformity sets [Table 1].

Anatomy

Tibialis posterior originates from the posterolateral tibia, posteromedial fibula and interosseous membrane. It runs through the deep posterior compartment of the leg and its tendon passes behind the medial malleolus. Blood supply to the tendon is poorest in this area and is the most common site for rupture.[3]

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Close to its insertion site the tendon splits into a main, plantar and recurrent components, with the main component inserting onto the navicular tuberosity, the plantar portion onto the second, third, fourth metatarsals, second and third cuneiforms and cuboid. The recurrent component attaches to the sustentaculum tali of the calcaneus.[4]

Tibialis posterior acts as the primary dynamic stabilizer of the medial longitudinal arch and main inverter of the midfoot.[5,6] Its contraction also elevates the medial longitudinal arch, causing the hind foot and midfoot to become a rigid structure. This allows the gastrocnemius to act with greater efficiency during the gait cycle.[5]

Pathophysiology

Acute/traumatic rupture of the posterior tibial tendon is the cause of acquired flatfoot and may result from ankle fractures or direct trauma to the tendon.[7,8] A complete rupture of the tendon is not required for development of adult flatfoot; due to the short excursion of the tendon, lesser damage may render it ineffective. The more usual cause is tendinosis from repeated microtrauma. As the tendon degenerates, it is replaced with ineffective fibrotic tissue.[9] This may occur in a zone of hypovascularity in the region of the medial malleolus.[3,10]

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As the tendon loses function, the medial longitudinal arch of the foot collapses, which causes a relative internal rotation of the tibia and talus.[11] There is eversion of the subtalar joint, which forces the heel into valgus alignment, and abduction at the talonavicular joint. Varus alignment of the heel causes a lateral shift in the normal axis of the achilles tendon, which in time will lead to a contracture.[12,13] As the deformity gets worse, the distal fibula comes into contact with the lateral calcaneus, causing lateral hindfoot pain.[5]

Epidemiology

Posterior tibial tendon dysfunction typically occurs in obese, middle-aged women[5,14-17] with up to 10% prevalence in this group.[18] Conditions such as diabetes, hypertension, obesity, previous surgery, foot/ankle trauma and steroid use is found in up to 60% of patients.[4] Seronegative spondylarthropathies have also been identified as risk factors,[11] as have local steroid injections.[4,19] Though many of these risk factors are generalized medical conditions, only one side is typically affected; bilateral disease is rare [Table 2].[20]

Classification

Johnson and Strom developed a classification scheme in 1989 to describe the stages of posterior tibial tendon dysfunction.[15] Originally three stages were described, with Stage I having an intact tendon with inflammation, but no clinical deformity. Treatment at this stage is conservative, with tenosynovectomy and debridement for severe cases.[10,21] In Stage II the tendon is ruptured or otherwise nonfunctional, resulting in a planovalgus deformity which is passively correctable. In Stage III the foot deformity is no longer correctable and osteoarthritis is seen in the subtalar joint. A fourth stage, added by Myerson, describes patients with a valgus tilt of the talus in the ankle joint with tibiotalar degeneration.[5,15] Surgical correction of Stage III and IV involves fusion of one or more joints, including the subtalar, calcaneocuboid, talonavicular and ankle joints [Table 3].

History

Most patients report a slow, insidious onset of unilateral flatfoot deformity.[15,18] A history of trauma may be present in up to 50% of cases.[22] Patients will describe the pain and swelling along the medial aspect of the foot and ankle, which may be exacerbated with activity. Standing on their toes may be painful and difficult, as may walk up or down stairs or on uneven surfaces. In addition, patients may complain of an exacerbation of a preexisting limp. As the medial longitudinal arch collapses, the deformity of the foot increases; in this instance patients may describe abnormal wear on their shoes. In severe cases of deformity the distal fibula will come into contact with the calcaneus, and pain will move to the lateral aspect of the foot; patients at this stage may describe the feeling of walking on the medial ankle.[23]

Table 1: Posterior tibial tendon dysfunction-overview

Initial insidious course with nonspecific symptomatology: patients report pain in the medial hindfoot, difficulty walking on uneven surfaces
Typically unilateral acquired flatfoot deformity
Examination findings include flatfoot deformity, inability to perform single heel raise, and "too many toes" sign
Nonoperative management is favored in early stages and may halt progress of disease
Conservative treatment options include nonsteroidal anti-inflammatory drugs, rest and immobilization, orthotic devices
Surgical intervention is usually required in Stage II, III, and IV

Table 2: Risk factors for posterior tibial tendon dysfunction

Diabetes
Hypertension
Obesity
Steroid use/local steroid injection
Previous surgery
Foot/ankle trauma
Seronegative spondylarthropathies

Table 3: Posterior tibial tendon dysfunction-stages

I	Inflamed, intact tendon without clinical deformity
II	Ruptured or nun-functional tendon with planovalgus deformity
III	Advanced foot deformity with subtalar joint osteoarthritis
IV	Ankle joint involvement with tibio-talar degeneration

Examination

In Stage, I posterior tibial tendon dysfunction there is swelling and tenderness along the course of the tendon, typically behind and below the medial malleolus[1,5] accompanied by weakness with inversion of the foot.[5,23] As the dysfunction progresses, patients may have less swelling and pain but this alludes to a complete rupture of the posterior tibial tendon and impending flatfoot deformity.

Inspecting the patient from behind will show a valgus position of the heel and flattening of the medial longitudinal arch with compensatory forefoot abduction. A "too many toes sign" will be present where more than one to two toes are seen along the lateral aspect of the affected foot.[1,5]

One of the most sensitive tests for posterior tibial tendon dysfunction is the single limb heel rise. To perform this test, the patient uses their arms to balance themselves against the wall. The patient will then lift the opposite foot off the ground and attempt to rise onto the toes of the affected foot.

When performing a single heel raise patients may have difficulty performing a single heel raise or weakness after multiple heel attempts.[1,5] Patients with a fully functional tendon can complete 8–10 repetitions, but by Stage II, the vast majority of patients are unable to perform a single unsupported heel rise (or may only be able to complete a few).

An attempt to correct any valgus deformity of the hind foot should be made by grasping the heel. In Stage II the deformity will still be correctable but by Stage III and IV it will not be reducible.

Investigations

Weight bearing anteroposterior and lateral views of both feet, in addition to bilateral weight bearing ankle views, should be obtained. Changes may not be apparent on X-ray in the early stages but as the disease progresses, collapse of the medial longitudinal arch and joint degeneration may become evident.[15,24,25]

Ultrasound is a quick, readily available, low-cost investigation which gives information about tendon size, degeneration and presence of fluid. Quality of results, however, can be ultrasonographer dependent.[25-27]

Magnetic resonance imaging (MRI) is useful in detecting early changes within the tendon and for assessing joint degeneration in later disease.[25,28-30] Detecting early tendon changes and prompt initiation of nonoperative treatment may avert the need for surgery. MRI is also useful in more advanced stages of the disease, where precise delineation of joint degeneration plays a part in preoperative planning.

Treatment

Management of posterior tibial tendon dysfunction relies upon accurate staging of the disease, as both nonoperative and operative treatment options change with each disease stage. Aims of treatment are to relieve pain, improve function, restore alignment by correction of any deformity, and to halt or slow progression of the disease. We recommend early referral to an orthopedic surgeon.

Stage I disease

A walking cast or CAM boot can be used to immobilize the foot. If this brings relief, the patient can have shoe inserts or modifications, orthotics or an ankle-foot orthosis (AFO) fitted. Options for custom orthotics include the University of California Biomechanics Laboratory Orthosis, an AFO, or a removable boot. Orthotics aims to provide arch support and correct the flexible component of the deformity.[31,25] Physiotherapy for achilles tendon stretching and tibialis posterior strengthening along with nonsteroidal anti-inflammatories can also help.[32-34] Up to 4 months of nonoperative treatment should be trialed; if there is no improvement after this period, a tendon synovectomy or debridement may be indicated.[2]

Stage II disease

Nonoperative methods can still be trialed at this stage and include walking cast, CAM boots, or orthotics. These interventions often prove to be ineffective and surgical treatment is typically carried out in Stage II of the disease. This is because the deformity is still correctable and joint degeneration has not occurred, allowing for better joint alignment and possibly avoiding joint fusion. A variety of methods have been described, and these normally include bony procedures along with soft tissue reconstruction and tendon transfers.[12,13,16] Traditionally, this would consist of a combination of calcaneal osteotomy, posterior tibial tendon excision, flexor digitorum longus transfer, and achilles tendon lengthening. These aim to correct the flexible deformity and dynamically stabilize the medial arch.[16,21,22,33]

Stage III and stage IV

At Stage III the deformity is rigid and at Stage IV there is ankle joint involvement.[5] Nonoperative treatment at this stage consists of specialized shoes, orthotics, AFO, and inserts that accommodate the deformity. The collective primary aim is analgesia; secondarily they may serve to slow progression of the disease. Surgical intervention at these stages will involve multiple bony procedures such as osteotomies and arthrodesis. In Stage III typically a triple arthrodesis (fusion) of the calcaneocuboid, talonavicular and subtalar joints is undertaken, whereas in Stage IV an ankle joint fusion is performed, in addition to a triple arthrodesis.[5,12,16,35,36]

Conclusion

Posterior tibial tendon dysfunction characteristically is a slow onset condition mainly affecting middle-aged, obese women. Risk factors include obesity, hypertension, diabetes, steroid use and seronegative arthropathies. Patients may complain of pain and swelling around the medial ankle, difficulty mobilizing or exacerbation of an existing limp. Examination may show tenderness along the course of the tendon, difficulty performing a single heel raise or "too many toes" when feet are viewed standing from behind. X-ray and MRI can confirm diagnosis, help stage disease and assist in preoperative planning. The disease as an entity is under-recognized, and early stages of the disease can be misdiagnosed, but prompt treatment can prevent deformity and need for surgery.

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